Migrating Grass Awns in a Dog with Pericardial Effusion

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INTRODUCTION

Grass awns are common causes of soft tissue infections, abscessation, and draining tracts in animals.[1] Because of the angled shape of their barbs, grass awns can unilaterally move through tissue, such as between fascial planes and along airways or the esophagus.[2] Migrating foreign bodies (FBs) can cause a wide array of clinical signs that vary depending on their location.[1] Septic pericardial effusion is a rare but serious sequela to migrating FBs and can result in acute life-threatening conditions such as cardiac tamponade, pericarditis, epicarditis, and right-sided congestive heart failure signs.[3,4] Right-sided congestive heart failure signs occur secondary to right atrial collapse, pericardial effusion, or constrictive epicarditis. Current treatment recommendations include surgical debridement of infected tissue and antibiotic therapy.[1,4-6] Grass awns are often challenging to locate during surgery. Imaging modalities such as preoperative and intraoperative transthoracic echocardiography (TTE), computed tomography (CT), and magnetic resonance imaging (MRI) have been used to locate small FBs and avoid radical, excessive dissection.[7,8] One recent study showed that intraoperative ultrasound improved the rate of localization and removal of FBs compared with preoperative ultrasound, CT, or MRI.[8]

This report describes the successful localization and removal of five grass awns embedded within the pericardium of a dog with septic pericardial effusion, pericarditis, and epicarditis on the basis of preoperative TTE alone; no advanced diagnostic imaging was needed.

CASE PRESENTATION

A 5-year-old male, neutered chocolate Labrador retriever (34 kg) was presented for further assessment of previously diagnosed pericardial, pleural, and abdominal effusions made by the referring veterinarian.

Approximately 6 weeks before the dog's presentation, it became acutely lethargic and anorexic and was presented to the referring veterinarian. Thoracic radiographs showed cardiac silhouette enlargement, and point-of-care ultrasound (POCUS) showed pericardial effusion. POCUS examinations are typically very limited studies that assess solely for the presence or absence of effusions (i.e., pericardial, pleural, and abdominal) and they are not complete transthoracic echocardiographic studies. Pericardiocentesis removed 700 mL of clear to mildly hemorrhagic fluid. In-house cytology of the fluid showed no evidence of neoplasia, and no further description of the cytology was provided. Approximately 9 days later, the patient was brought back to the referring veterinarian for suspected new effusion observed in the abdomen. Pericardial effusion was found to have recurred, via POCUS, and pericardiocentesis was again performed, without reporting the quantity of the effusion. Cytology of the pericardial fluid showed increased leukocyte count but no evidence of neoplasia, with no further description of the cytology. Culture of the pericardial effusion was negative. Thoracic POCUS showed a small volume of pleural effusion and lung atelectasis. Following pericardiocentesis, scant pericardial effusion was present on POCUS. The patient was discharged from the referring veterinarian with a diagnosis of suspected idiopathic pericardial effusion.

The following day, the patient was found laterally recumbent after vomiting at home. The dog was brought emergently to the referring veterinarian and was fluid resuscitated for shock. POCUS revealed recurrence of pericardial, pleural, and abdominal effusions. Once the dog was stable, effusion was again removed from the pericardial space, via the use of POCUS. The patient was stabilized and discharged on doxycycline 400 mg (11 mg/kg) orally every 12 hours, enrofloxacin 136 mg (4 mg/kg) orally every 12 hours, firocoxib 113.5 mg (3.3 mg/kg) orally once daily, furosemide (unknown dose), and a probiotic orally once daily. For 2 weeks following this visit, the patient remained stable but persistently tachycardic (140-150 beats/min) and tachypneic (30-40 breaths/min) with increased respiratory effort at home.

The patient was then referred to the University of Wisconsin Small Animal Internal Medicine service for further workup of the effusions. On presentation, the patient was euhydrated and in good body condition (body condition score 6 of 9), the heart rate was 132 beats/min, and tachypnea was present (42 breaths/min). Heart sounds were reduced bilaterally, with a regular rhythm and normal pulse quality. POCUS revealed pericardial effusion with collapse of the right atrium with mild cranial pocketing of pleural effusion and a moderate amount of fibrin-like material in the pleural cavity. Moderate peritoneal effusion was also present. A therapeutic pericardiocentesis was attempted, via POCUS, but a laceration was created in the pericardium, leading to uncertainty of the effusion origin (pleural vs pericardial). Cytologic examination of effusion revealed a low-protein fluid with a component of hemorrhage. Analysis of the peritoneal effusion showed a high-protein transudate. There was no evidence of overt infectious disease or neoplasia in these samples.

To further investigate the cause for pericardial effusion and collapse of the right atrium, the patient was then transferred to the cardiology specialty service. TTE showed moderate, generalized pericardial effusion. Careful evaluation of the pericardium revealed a focal region at the apex of the left and right ventricles to slightly lateral on the left

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE, right parasternal long-axis four-chamber view. Pericardial effusion can be seen at the *top* and *bottom* of the image. The right atrium is collapsed in diastole and systole, with no right auricular appendage seen. The left atrium and left ventricular (LV) dimension are normal, with normal LV function, normal mitral, and tricuspid valve leaflets.

Video 2: Two-dimensional TTE, left parasternal long-axis four-chamber view, demonstrates that the pericardium is adhered to the epicardium at the apex, where a hyperechoic lesion is seen, suspected to be an FB. Pericardial effusion can be seen on the *right* and *left* of the image. The right atrium is collapsed in diastole and systole, with no right auricular appendage seen. Left atrial and left ventricular size and function, mitral valve leaflets, and tricuspid valve leaflets are all normal.

Video 3: Two-dimensional TTE, obliqued right parasternal long-axis four-chamber view, including the left ventricular outflow tract, demonstrates that the right atrium is collapsed in diastole and systole, with no right auricular appendage seen. Pericardial effusion can be seen at the *top* and *bottom* of the image; aortic leaflets are normal.

Video 4: Two-dimensional TTE, obliqued right parasternal short-axis view of the heart base, demonstrates that the right atrium is collapsed in diastole and systole, with no right auricular appendage seen. Pericardial effusion can be seen at the *top left* and *bottom right* of the image. Pleural effusion is also present at the *top left* of the image; tricuspid valve leaflets are normal.

Video 5: Two-dimensional TTE, obliqued left parasternal long-axis four-chamber view, demonstrates that the right atrium is collapsed in diastole and systole, with no right auricular appendage seen. Pericardial effusion can be seen at the *left* and *right* of the image; tricuspid valve leaflets are normal.

Video 6: Median sternotomy surgical approach (head at the *top* of the image, left thorax on the *right* of the image) demonstrates the fibrous and roughened cardiac appearance, consistent with epicarditis, after pericardiectomy. The remaining pericardium at the base of the heart is severely thickened, consistent with pericarditis.

Video 7: Two-dimensional TTE, right parasternal long-axis, four-chamber view, demonstrates better filling of the cardiac chambers after pericardiectomy. Pleural effusion can be seen at the *left top* and *left bottom* of the image; mitral and tricuspid valve leaflets are normal.

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ventricular posterior wall where the pericardium appeared adhered to the epicardium (Figures 1 and 2, Video 1). On the left parasternal views, there was a shadowing, hyperechoic lesion at the left and right ventricular apex (Figure 2, Video 2). This area of the pericardium also appeared to be adhered to the epicardium. There was diastolic and systolic collapse of the right atrium, and the right auricular appendage could not be identified (Figure 3, Videos 3-5).

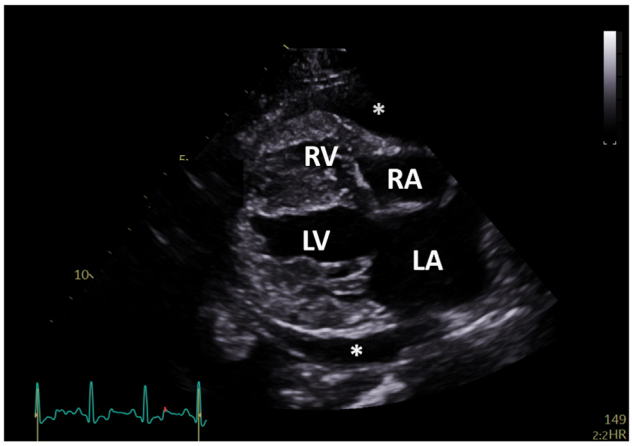

Figure 1 Pericardial effusion. Two-dimensional TTE, right parasternal long-axis four-chamber view, demonstrates pericardial effusion (*white asterisks*). *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

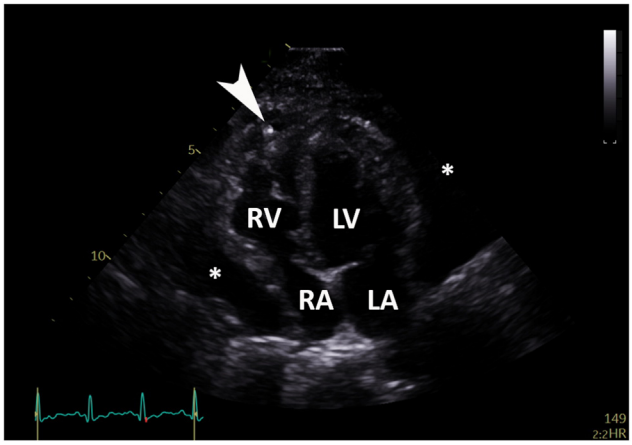

Figure 2 Adhered apical pericardium and FB. Two-dimensional TTE, left parasternal long-axis four-chamber view, demonstrates pericardial effusion (*white asterisks*). The *white arrowhead* indicates where the pericardium is adhered to the epicardium and a hyperechoic lesion is seen, suspected to be an FB. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

The final diagnosis, on the basis of TTE performed by a board-certified veterinary cardiologist, was presumptive septic pericarditis secondary to an FB with subsequent pleural and abdominal effusions as a result of right atrial collapse. Epicarditis and pericarditis were also hypothesized to be present because the right auricular appendage could not be seen because of adherence of the right auricular appendage and right atrium from fibrous adhesions. Because of the strong suspicion for septic pericarditis, epicarditis, and pericarditis secondary to an FB within the pericardium, urgent thoracic exploratory surgery and subtotal pericardiectomy were recommended.

Thoracic exploration through a median sternotomy was performed the next day. The lungs were noted to be adhered to the parietal pleura upon entrance along midline. The pericardium was noted to be irregularly thickened, with abnormal adhesions to the sternum. At the apex of the heart, the pericardium was indiscernible from the epicardium, and there was no space between the pericardium and the epicardium because of severe adhesions. The pericardium was

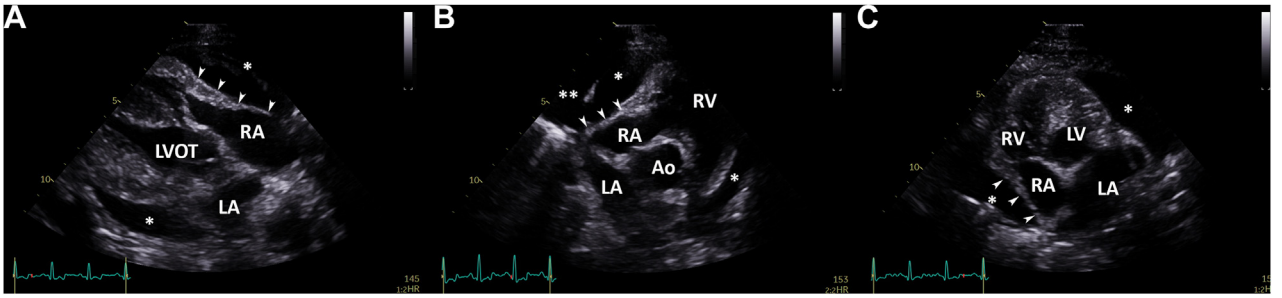

Figure 3 Diastolic and systolic collapse of right atrial wall and inability to see the right auricular appendage. Two-dimensional TTE, obliqued right parasternal long-axis four-chamber view **(A)**, obliqued right parasternal short-axis view of the heart base **(B)**, and obliqued left parasternal long-axis four-chamber view **(C)**, demonstrates pericardial effusion (*white asterisks*). *White arrowheads* indicate the diastolic and systolic collapse of the right atrial wall and the absence of the right auricle. *Ao*, Aorta; *LA*, Left atrium; *LV*, left ventricle; *LVOT*, left ventricular outflow tract; *RA*, right atrium; *RV*, right ventricle.

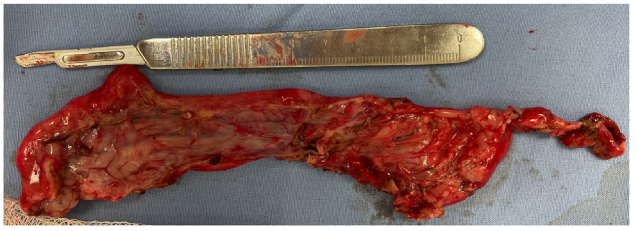

Figure 4 Pericarditis. Gross pathology of the excised pericardium. The pericardium is severely thickened. The tissue is light tan to dark brown, soft with multifocal firm to hard, gritty regions, and has an irregular surface.

incised and dissected from the epicardial surface over the cardiac apex using a combination of blunt and sharp dissection. The dissection was extended to the dorsal aspect of the heart. A moderate amount of serosanguinous fluid was removed from cavitated areas within the pericardium using suction. The pericardium overlying the right auricular appendage and atrium was not removed because of extensive adhesions to the epicardium. The right auricular appendage appeared to be adhered to the right atrium, and because of concern for lacerating the right auricular appendage or right atrium, further dissection to release the auricular appendage from the atrium was not pursued. Following subtotal pericardiectomy, the removed tissue was inspected for foreign material (Figure 4).

Five grass awns were identified and isolated from the fibrinous pericardial tissue at the level of the left and right ventricular apices, confirming the diagnosis by TTE (Figure 5).

The thoracic cavity was then inspected before closure. The epicardium was rough and irregular in texture, with evidence of fibrosis, consistent with epicarditis (Video 6). The chest was flushed copiously with sterile saline. The pericardium was submitted for histopathology in addition to aerobic and anaerobic culture and sensitivity. The thoracotomy was closed routinely.

Histopathology of the excised pericardium showed severe, multifocal to coalescing, chronic-active pyogranulomatous, lymphoplasmacytic, and proliferative pericarditis with fibroplasia and fibrosis, mesothelial hyperplasia, intralesional plant material and gram-negative coccobacilli (Figure 6).

A sternal lymph node was also submitted and showed severe lymphoid hyperplasia and sinus histiocytosis with draining edema, hemorrhage, and neutrophils. Aerobic and anaerobic cultures of pericardium grew *Actinomyces canis* (aerobic) and *Peptostreptococcus canis*

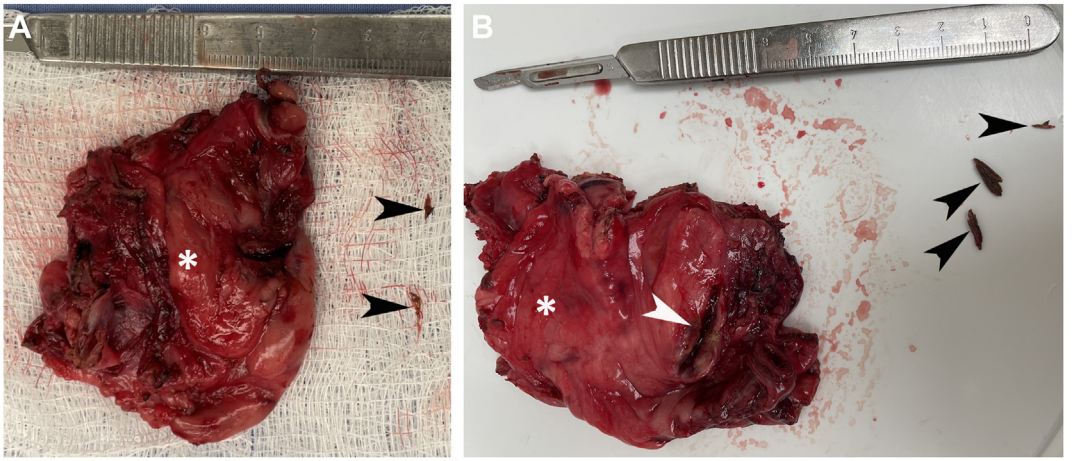

Figure 5 Grass awns Gross pathology of the severely thickened, excised pericardium from the apex of the heart (*white asterisks*). *Black arrow heads* indicate grass awns found in the pericardium **(A** and **B)**. *White arrowhead* in B shows an excised nodule that contained a grass awn.

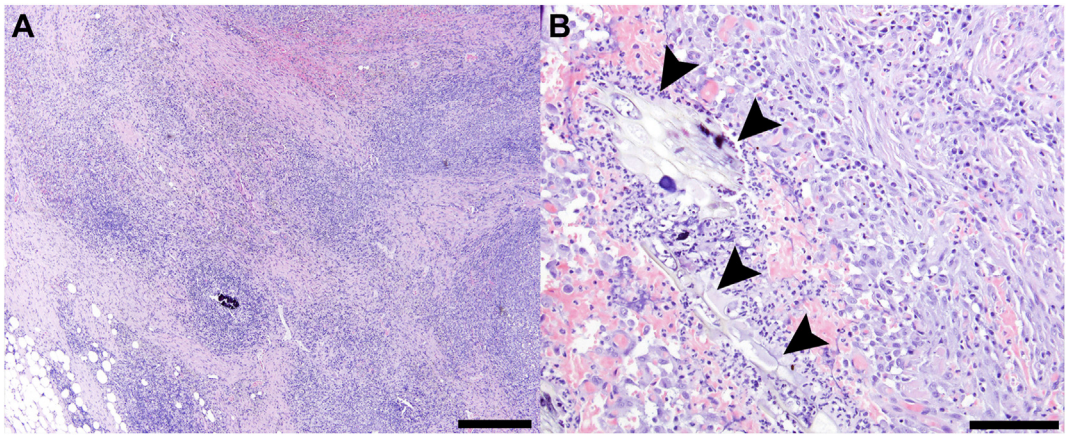

Figure 6 Pericarditis and grass awn histopathology. Histologic images of the pericardium, hematoxylin and eosin stain. **(A)** The pericardium is markedly thickened by fibrosis, occasional foci of mineralization, and large nodular aggregates of inflammation composed of macrophages, neutrophils, lymphocytes, and plasma cells. Scale bar, 200 μm. **(B)** Rare fragments of plant material (*arrowheads*) are found within the inflammatory foci. Scale bar, 50 μm.

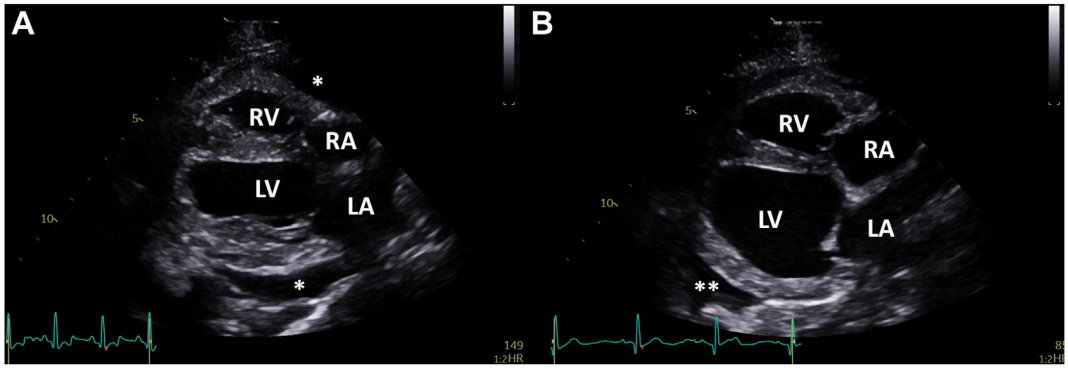

Figure 7 Improved chamber filling before and after pericardiectomy. Two-dimensional TTE, right parasternal long-axis four-chamber view. Prepericardiectomy TTE **(A)** demonstrates diminished chamber sizes compared with postpericardiectomy TTE **(B)**. *Single white asterisks* indicate pericardial effusion. *Double white asterisks* indicate pleural effusion. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

and *Prevotella heparinolytica* (beta lactamase negative; anaerobic). The antibiotic enrofloxacin (408 mg orally every 24 hours for 10 days) was prescribed at discharge.

TTE was performed 1 day postoperatively and showed significant improvement in four-chamber filling. There was no evidence of remaining foreign material (Figure 7, Video 7). The owners of the patient were contacted 8 months following surgery. The dog was alive, with no clinical signs suggestive of recurrent effusion appreciated at home, and had resumed its full activity level.

DISCUSSION

Pericardial effusion and cardiac tamponade secondary to septic pericarditis are a rare cause of acquired cardiovascular disease in dogs and cats.[1-5,9,10] Pericardial effusion more often occurs secondary to either idiopathic or neoplastic processes (e.g., hemangiosarcoma, heart base tumors), and presentations can appear clinically similar.[5,6] In the patient presented in this report, a neoplastic or an idiopathic process were initially considered as top differential diagnoses for the patient's effusions for several reasons. First, the chronic nature of recurrent effusion was more suggestive of either a neoplastic or an idiopathic process rather than an infectious process. Additionally, in the face of previous negative cultures and no bacterial growth on pericardial fluid, an infectious process was lower on the list of differential diagnoses. Presenting clinical signs associated with pericardial effusion secondary to FB migration are often vague until they become severe enough to cause conditions such as cardiac tamponade and right-sided congestive heart failure signs. Consistent with reports from similar patients, the lack of a positive culture of pericardial fluid may be attributed to administration of antibiotics throughout the chronic course of the animal's illness before presentation, and thus, neutrophilic pericardial effusion was not diagnostic for a septic process in advance of surgery.[4]

Diagnosis of pericardial FBs as a cause of septic pericarditis and pericardial effusion can be technically challenging. Success using a

combination of transthoracic, transesophageal, and intraoperative echocardiography has been described.[7,9] A recent study showed that intraoperative ultrasound improved the rate of localization and removal of FBs compared with CT and MRI.[8]

The key findings on TTE indicating pericarditis in this patient included a hyperechoic lesion that shadowed at the apex of the heart, pericardial adherence to the epicardium, and a diastolic and systolic collapse of the right atrium with an indiscernible right auricular appendage, suggesting fibrous adhesions from epicarditis. Doppler studies assessing for diastolic dysfunction as seen with constrictive disease (i.e., mitral inflow velocities, tissue Doppler imaging of the left and right ventricles) of the heart were performed, with normal findings.

This report describes preoperative TTE as a noninvasive and accurate way to identify a cause for presumed idiopathic pericardial effusion, inform a diagnosis, and aid in surgical planning.

CONCLUSION

This patient highlights the utility and sensitivity of preoperative TTE in determining a cause of pericardial effusion of unknown etiology. This noninvasive imaging modality was able to accurately identify the presence and approximate location of an FB within the pericardium. Often, more advanced diagnostic imaging (i.e., CT or MRI) is needed to identify the presence of an FB, epicarditis, and pericarditis. Complete and careful TTE needs to be performed by a skilled echocardiographer when trying to identify an FB as the cause of pericardial effusion and to scrutinize for structures and function that might lead to a diagnosis of epicarditis and pericarditis, as these are often life-threatening sequelae. Recommendations for animals with idiopathic pericardial effusion should be complete TTE by a skilled echocardiographer before a diagnosis of idiopathic effusion, especially if the pericardial effusion continues to reoccur. The present patient also highlights the importance of echocardiographic imaging and the translation to the pathologic findings.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with the following guidelines: research animal resources and compliance policy at the University of Wisconsin–Madison.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

FUNDING STATEMENT

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DISCLOSURE STATEMENT

The authors report no conflict of interest.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2023.11.002.

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